Severe intestinal bleeding due to sinistral portal hypertension after pylorus-preserving pancreatoduodenectomy

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Severe intestinal bleeding due to sinistral portal hypertension after pylorus preserving pancreatoduodenectomy

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Running Title: Sinistral portal hypertension

Abstract

A rare case of severe intestinal bleeding caused by sinistral portal hypertension after pylorus preserving pancreatoduodenectomy is reported. Some examinations revealed that the developed collateral vessels adjacent to the pancreas and attollens jejunum in the right upper abdomen were the source of the intestinal bleeding, and typical gastric varices were not identified. The present case showed atypical development of collateral vessels due to the postoperative state, whith interfered with the making of a diagnosis.

Keywords: Sinistral portal hypertension; intestinal bleeding; splenomegaly; gastric varices

Sinistral, or left-sided, portal hypertension is a rare cause of upper gastrointestinal hemorrhage [1–5]. Typically, compression of the splenic vein causes backpressure in the left portal venous system and subsequent gastric varices [1]. However, in the postoperative state, the pathophysiologic condition is altered making it difficult to diagnose. We present here a case of atypical sinistral portal hypertension whose pathophysiologic condition was complicated because of the postoperative state.

Case report

A 53-year-old man with severe intestinal bleeding was referred to our hospital for examination. A pylorus preserving pancreatoduodenectomy (PpPD) with resection and reconstruction of the superior mesenteric artery (SMA) and portal vein (PV) had been performed for a mesenteric tumor approximately four years rprevirouly previously. It turned out to be an inflammatory myofibroblastic tumor of the mesentery, although a malignant tumor had been suspected on pre- and intraoperative examinations. Recently, he had been discharging blood for a month and presented with anemia (Hb 7.3 g/dL; normal range 11.2–14.5 g/dL, red blood cell $229\times10^4/\mu L$: normal range $430–550\times10^4$, Ht 22.2%; normal range 39.7–51.2, platelet 12.6×10^4; normal range 13–35). The serum levels of liver enzymes were normal. The other laboratory data were within the respective normal ranges. The cause was not identified by any examinations including upper

and lower gastrointestinal tract endoscopy and small-bowel endoscopy. During the examination period, the anemia rapidly progressed and numerous blood transfusions (32 units per 5 days) were needed. One day, he developed massive melena, resulting in hypotension. An emergent angiography was performed to determine the cause and treat the bleeding.

No active arterial bleeding was identified on celiac or superior mesenteric arteriograms. The reconstructed SMA and PV were demonstrated without failure or obstruction (Figure. 1). On the arterial phase of CT during celiac and superior mesenteric arteriography, no active arterial bleeding was identified. Portal venous phase CT showed the ligated splenic vein without reconstruction. The flow of the splenic vein stopped at the ligated portion, and drained into PV via mainly Henle's gastrocolic trunk and coronary vein. The posterior and short gastric veins were also slightly dilated. This atypical drainage formed the developed collateral vessels mainly adjacent to the pancreas and attollens jejunum in the right upper abdomen (Figure. 2). No typical gastric varices were identified. In addition, the spleen was enlarged as compared to four years earlier (before operation), and the liver had not become deformed. Continuously, bleeding scintigraphy with Tc-99m DTPA human serum albumin (Tc-99m HSA-DTPA) was performed and showed uptake in the right upper abdomen. This was consistent with the bleeding from the developed collateral vessels noted on CT.

In this way, it was elucidated that this condition with a ligated splenic vein without reconstruction showed the same pathophysiologic features as those

of so-called sinistral portal hypertension resulting in severe intestinal bleeding. Splenectomy was performed immediately. Subsequently, the melena and progressive anemia disappeared, and the Hb/Hct improved. CT one month after operation showed reduction in the size of the collateral vessels.

Discussion

Sinistral (left-sided) portal hypertension was first reported by Greenwald and Wasch in 1939 [6]. It is a localized form of portal hypertension that occurs as a result of isolated thrombosis or obstruction of the splenic vein [7, 8]. The features distinguishing it from other forms of portal hypertension are preserved liver function and a patent extrahepatic portal vein [1].

Most patients with left sided portal hypertension are asymptomatic. In symptomatic patients, the first clinical manifestation is generally acute or chronic upper gastrointestinal hemorrhage from gastric varices. Anemia due to hypersplenism or upper gastrointestinal hemorrhage is also identified. Abdominal pain without bleeding sometimes occurs [1].

The main cause of sinistral portal hypertension is pancreatic disorders because of the splenic vein's location. Chronic pancreatitis is the most common cause [8], and any type of pancreatic disease is likely to involve the splenic vein [1, 9–12]. Various disorders other than pancreatic diseases can also cause splenic vein thrombosis/obstruction, although they are rare [1, 13–15].

Following splenic vein thrombosis/obstruction, splenic blood typically drains through the short or retro gastric veins to the stomach. In the gastric wall veins of the fundus, blood flow and pressure increase and submucosal

structures consequently dilate, producing gastric varices and often, but not always, esophageal varices [1, 16]. However, due to several anatomic variations, obstruction of the splenic vein may not always result in portal hypertension or formation of varices. These several anatomic variations effect the formation of varices.

In the present case, when PpPD was performed, the surgeon intentionally did not make an anastomosis between the reconstructed portal vein and ligated splenic vein, because a prognostically unfavorable malignant tumor had been suspected. However, four years after the operation, the ligated splenic vein caused varices to develop in an atypical region that was adjacent to the pancreas and attollens jejunum in the right upper abdomen. The formation of gastric varices or gastrointestinal bleeding sometimes occurs after surgical procedures without a reconstructed splenic vein [17, 18]. On the other hand, some surgeons reported that reconstruction of the splenic vein is not necessarily required, especially when the left gastric vein and IMV are preserved [19, 20]. This is an atypical case in which varices developed adjacent to the pancreas and attollens jejunum in a sinistral portal hypertension post operative state, and the reason why typical gastric varices did not form despite the existence of the short and posterior gastric veins is unknown.

Management is usually surgical to treat the underlying pathology and splenectomy to decompress the left portal venous system [4, 5].

Splenectomy decreases the arterial inflow into the left portal system by ligation of the splenic artery, resulting in decompression of the (gastric) varices. In the present case, the melena and anemia improved immediately after splenectomy. Splenic artery embolization has also been suggested as an alternative to splenectomy in high-risk patients [5, 21] or as a preoperative measure to reduce intraoperative blood loss [22]. The overall prognosis for patients with sinistral portal hypertension is clearly dependant on the primary pathology [23]. Our patient has been doing well and has not developed melena or anemia in the year after splenectomy.

In conclusion, we presented a case of severe intestinal bleeding caused by atypical sinistral portal hypertension. The pathophysiologic condition was complicated because of the postoperative state. However, we should be familiar with the typical condition of sinistral portal hypertension and comprehend the present atypical condition even in emergencies.

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Figure legends

Figure 1 No active arterial bleeding is identified on arteriogram. The reconstructed superior mesenteric artery was demonstrated without failure or obstruction.

Figure 2

(A) (B) On axial CT during celiac arteriography, the flow of splenic vein stops at the ligated portion (black arrow), and drains into the portal vein (PV) (black arrowhead) via mainly Henle's gastrocolic trunk. This atypical drainage forms the developed collateral vessels adjacent to the pancreas and attollens jejunum in the right upper abdomen (white arrow). Coronary vein is also dilated (white arrowhead).

(C) On coronal maximum intensity projection, the atypical drainage forms the developed collateral vessels adjacent to the pancreas and attollens jejunum in the right upper abdomen (white arrowheads).